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VASCULAR DISEASE

LACTATE DEHYDROGENASE (LDH) AND RESPONSE TO RENAL ARTERY STENTING.

ACC Poster Contributions
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Background: Lactic acid dehydrogenase (LDH) is a marker of cell injury or death that increases with renal infarction; however, the utility for detecting renal injury, before, during or after renal artery stenting (RAS) has not been studied.

Methods: One hundred patients undergoing RAS at 7 centers were randomized to embolic protection or double-blind use of a GPIIbIIIa inhibitor in a 2×2 design. All data were analyzed blindly at independent core labs. GFR change was classified as improved (>20%), stable (-20% - +20%), or worsened (<20%).

Results: There was a significant negative correlation between baseline LDH and baseline GFR (-0.242, $p<0.05$) that persisted at 24 hours as well as 1-month(Figure 1). Following RAS LDH decreased at 24-hours (190±58 to 177±50, $p<0.05$) but increased at 1-month follow-up (213±60). Improved GFR 1-month following RAS was associated with high LDH ($p=0.02$)(Figure2,). In contrast worsened GFR after RAS was associated with lower initial LDH values that rose significantly at 1-month follow-up (+36, $p=0.006$) (Figure2,).

Conclusions: In atherosclerotic renal stenosis high baseline LDH is associated with low baseline GFR but improvement in GFR 1- month post procedure, potentially indicating ongoing renal injury amenable to treatment with stenting. In contrast, in patients with worsened GFR 1- month post-procedure, LDH rises significantly suggesting ongoing injury late after the procedure.